Reply

Rittoo and Gamra et al. make some interesting observations on the problem of two jets of mitral regurgitation after balloon mitral valvuloplasty. As mentioned in our study in *JACC*, the explanation for this phenomenon is uncertain. It is quite likely that, when commissures are split during balloon mitral valvuloplasty, the leaflets may develop localized incompetence as a result of failure of satisfactory coaptation of the leaflets or, equally possibly, as a result of some small degree of leaflet tearing. The observations of Rittoo and his colleagues in this regard, using transthoracic echocardiography and biplane transesophageal echocardiography, are interesting and we await formal publication of their work.

Gamra et al. report similar observations in "several patients," suggesting that the extra, peripheral jets of mitral regurgitation can be seen to arise from the base of the mitral valve leaflet at the site of commissural splitting, in close proximity to the anulus. Again, this explanation is compatible with commissural splitting or, equally likely, with associated tearing of the mitral leaflet in this region. It would seem that echocardiography early after valvuloplasty would be the optimal technique to assess this problem, and further study of this question would be appropriate.

When our observations were made, these phenomena were certainly unusual and not previously described. It is now clear, however, that they are more common than previously realized, presumably as a result of more widespread application of the technique of mitral balloon valvuloplasty and close echocardiographic monitoring after valvuloplasty.

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Meta-Analyses of the Effects of Thrombolysis and Aspirin on Clinical Outcomes

We read with interest both the careful meta-analysis by Roux et al. (1), addressing the effects of aspirin on reocclusion and recurrent ischemia after thrombolysis, and the accompanying editorial comment (2). Their analysis adds biologic plausibility to the previously reported findings of synergy between aspirin and thrombolysis (recombinant tissue-type plasminogen activator [rt-PA] or streptokinase) as determined through a meta-analysis of all randomized clinical trials assessing mortality as the primary clinical trial outcome (3,4). Examining randomized controlled clinical trials and their primary end point is more straightforward than the difficult task completed by Roux et al. The dose of rt-PA was relatively uniform in the clinical trials and was not confounded with aspirin administration. There is no controversy about death as an outcome. Trials in which thrombolysis (with or without aspirin) was confounded with further therapy (for example, coronary angioplasty) could be—and were—excluded from the analysis. In addition, since the randomized clinical trials included a total of about 26,000 patients, it was not necessary to include weaker evidence from nonrandomized studies. In our analysis the decrease in the odds of death was 40% when thrombolysis and aspirin were compared with aspirin alone, and 24% when thrombolysis without aspirin was compared with no aspirin in a control group. These differences are statistically ($p < 0.02$) and clinically significant and help explain the apparently disparate results of some thrombolysis trials. The consistency of the results of these two meta-analyses assessing the effects of thrombolysis and aspirin on different clinical outcomes is encouraging.

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